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Oral Manifestations of Smoking

Dr. Subramony B[1], Dr. Bhumika Gumber[2], Dr. Gopikrishnan[3]

[1,2]MDS, Dept of Periodontics, PGIDS Rohtak
[3]MDS, Dept of Oral Pathology, PGIDS Rohtak

INTRODUCTION

The oral tissues of smokers are exposed to high nicotine concentrations that negatively affect local cell populations. Gingival crevicular fluid nicotine concentrations can be up to nearly 300 times that of nicotine plasma concentrations in smokers (20ng/ml). Levels of nicotine in the saliva of smokers have been found to range from 0.5 μM to 9.8 μM. Nicotine:

- Impair gingival blood flow and revascularization during soft and hard tissue wound healing (due to vasoconstrictive properties).
- Inhibited expression of various growth factors which may negatively impact bone healing.
- Synergistic effect on inflammatory mediator production when bacterial lipopolysaccharide is combined with nicotine.
- Alter attachment of fibroblast to root surface and decrease collagen production while increasing collagenase activity.

Multitude of studies investigated the association of smoking status with a variety of periodontal and oral hygiene parameters, these included plaque indices, gingival indices, probing depth, clinical attachment levels and radiographic alveolar bone levels.

1. SMOKING AND ORAL HYGIENE

Higher levels of debris is seen in smokers than non smokers.

It is attributed to

1. Personality traits leading to decreased oral hygiene habits.
2. Increased rate of plaque formation
3. Combination of two.

Plaque composition and plaque accumulation rate in healthy and gingivitis condition do not seem to be different in smokers as compared to non smokers.

2. CIGARETTE SMOKING AND GINGIVAL DISEASE

Smoking and Gingivitis

- Early epidemiologic studies reported that smokers were found to exhibit more severe gingivitis symptoms than non smokers. The general interpretation was simple and straightforward: smokers had less efficient oral hygiene behavior, more plaque and therefore more gingivitis.[1] first observed that smokers may present with lower level of gingival inflammation to a specific level of plaque than non smokers. A number of later studies by Preber H et al[2] Bergstrom J et al[3, 4]have confirmed that the gingivitis expression in response to dental plaque is modified by smoking**.** This was observed despite of the lack of significant differences in plaque composition in smokers and non smokers. Development of gingival inflammation in response to experimental plaque accumulation (experimental gingivitis) was less pronounced in smokers than in non-smokers. Thus it is interpreted that cigarette smoking is an environmental exposure able to modulate gingivitis expression in response to dental plaque.
- Gingival bleeding as well as vascular hyperemic reaction associated with plaque induced gingivitis is suppressed in smokers[5,6].

- Winkellhoff V et al (2001) found the only clinical difference between smokers and non smokers in untreated patient groups was the amount of supragingival plaque in the smokers. They found no difference in the mean bleeding index between smoker and non smoker patients. A lower gingival bleeding index has been described in smoker patients with periodontitis in comparison to non smoker patients.[7]

Smoking and Pigmentation

- Oral pigmentations are increased significantly in heavy smokers.
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  - Reports from Sweden, Germany and Japan have shown tobacco smoking to be the most common cause for mucosal pigmentation in light skinned adult populations.
  - In one investigation of more than 31,000 caucasians, 21.5% of tobacco smokers exhibited areas of melanin pigmentation compared with 3% among those not using tobacco.
  - A higher frequency is seen in females, and it has been suggested that female sex hormones exert a synergistic effect when combined with smoking.

Although any mucosal surface may be affected, smoker's melanosis most commonly affects:

- Anterior facial gingiva in cigarette smokers.
- Commissural and buccal mucosa in pipe smokers
- Hard palate in reverse smokers.

Area of pigmentation significantly increase during the first year of smoking and appear correlated to the number of cigarettes smoked each day.

Smoking and mucosal changes

- Tobacco smoking is associated with leukoplakia development.

  - More than 80% of patients with leukoplakia are smokers
  - Heavier smokers have greater number of lesions and larger lesions than do light smokers.
  - Leukoplakia lesions either disappear or become smaller with in first year of habit cessation.

- Nicotine stomatitis a common mucosal change of hard palate. Palatal mucosa becomes diffusely gray or white; numerous slightly elevated papules are noted, usually with punctate red centres. Such papules represent inflamed minor salivary glands and their ductal orifices and mucosa that covers the papules frequently appears whiter than the surrounding epithelium.

  - Although this lesion is a white keratotic change obviously associated with tobacco smoking, it does not have premalignant potential because it develops in response to heat rather than the chemicals in tobacco smoke. Since pipe smoking generates more heat on the palate, nicotine stomatitis has been associated most often with this habit. Hand rolled cigarettes and cigars are smoked with the lit end held with in the mouth. The "reverse smoking" habit produces a pronounced palatal keratosis or reverse smoker's palate.
  - Palate returns to normal, usually within 1 to 2 weeks of smoking cessation.

Cigarette Smoking and ANUG:

Pindborgin a series of studies starting in 1947 to 1951 determined that tobacco smoking was a factor in NUG and that with the increase in the use of tobacco there was an increase in frequency of NUG.

3. CIGARETTE SMOKING AND PERIODONTITIS

In Adults.

- Using criteria established by the Centers for Disease Control and Prevention (CDC)

  - Current smokers were defined as those that had smoked ≥ 100 cigarettes over their lifetime and smoked at the time of the interview

- Former smokers had smoked ≥100 cigarettes in their lifetime but were not currently smoking
- Nonsmokers had not smoked ≥ 100 cigarettes in their lifetime.

- The prevalence and severity of periodontal disease in former smokers is between that of nonsmokers and current smokers.
- Probing depth, clinical attachment loss, and alveolar bone loss have all been shown to be more prevalent and more severe among smokers as compared to non smoking controls
- An assessment of the relationship between cigarette smoking and periodontitis was performed in more than 12,000 dentate individuals over the age of 18 years as part of the National Health and Nutrition Examination Survey (NHANES III).Periodontitis was defined as ≥1 site with clinical attachment loss of ≥ 4 mm and pocket depth of ≥ 4 mm. Of the 12,000 individuals studied, 9.2% had periodontitis.
  - On average, smokers were 4 times as likely to have periodontitis as persons who had never smoked after adjusting for age, gender, race/ethnicity, education, and income/poverty ratio.
  - Former smokers were 1.68 times more likely to have periodontitis than persons who had never smoked.
- This study also demonstrated a dose-response relationship between cigarettes smoked per day and the odds of having periodontitis. In subjects smoking
  - ≤ 9 cigarettes per day, the odds for having periodontitis was 2.79
  - ≥ 31 cigarettes per day were nearly 6 times more likely to have periodontitis.

  With former smokers, the odds of having periodontitis declined with the number of years since quitting.
- The odds ratio for periodontitis in current smokers has been estimated to range from as low as 1.5 to as high as 7.3, depending on the observed severity of periodontitis. A meta analysis of six studies concluded that older adult smokers were nearly three times more likely to have severe periodontitis than nonsmokers.
- Smokers are 2.6 to 6 times more likely to develop periodontal disease than nonsmokers. The odds ratio for a moderate (15 to 30 pack years) smoker to have periodontal disease is 2.77 times that of a nonsmoker, and a heavy smoker (≥30 pack years) is 4.75 times more likely to have periodontal disease than a nonsmoker. Pack years can be defined as the number of cigarettes smoked per day multiplied by the number of years that an individual smoked.
- Over 10 years period bone loss has been reported to be twice as rapid in smokers when compared with nonsmokers. The prevalence of furcation involvement among smokers was high as was the degree of alveolar bone loss.
- The prevalence of moderate and severe periodontitis and % of teeth with more than 5mm attachment loss was more severe in current cigarette smokers, but cigar and pipe smokers showed the severity of disease intermediate between current cigarette smokers and nonsmokers or former smokers. Tooth loss is also increased in cigar and pipe smoker when compared with nonsmokers.

In young adults

- Smoking is more prevalent among young patients (≤ 35 years) with the generalized form of aggressive periodontitis compared to those with the localized form or with healthy periodontal tissues. Several studies have shown that compared to non-smokers, young adult smokers aged 19 to 30 years have a higher prevalence and severity of periodontitis, despite similar or lower plaque levels.
- Haber et al reported that the prevalence of periodontitis defined as having a site with attachment loss ≥2 mm and probing depth ≥ 4 mm, was three to four times higher in young smokers 19 to 30 years of age compared to non smokers.[8]

  The high "periodontal cost" of smoking has been calculated as 27 years of disease progression, in other words a 32- year old smoker has similar periodontal attachment loss as a 59-year old non-smoker.
- Relationship between smoking and destructive periodontal disease is generally considered to be biologically plausible.

  1. Smokers display higher levels of infection with causative microorganisms.
  2. Agents contained in tobacco smoke can modulate the inflammatory and immune responses. Eradication of smoking at the population level could result in decrease of 1% to 2% in the prevalence of severe destructive periodontal diseases.

Haber, 1994 has described a discrete, smoking – specific disease entity – Smoking associated periodontitis – is characterized by[8]

- Fibrotic gingiva with thickened margins
- Limited gingival redness and edema relative to disease severity
- Proportionally greater pocketing in anterior and maxillary lingual sites

Gingival recession at anterior sites and a lack of association between periodontal status and the level of oral hygiene.

REFERENCES

[1] Bergström J, Preber H. The influence of cigarette smoking on the development of experimental gingivitis. Journal of periodontal research. 1986 Nov;21(6):668-76.
[2] Preber H, Bergström J. Occurrence of gingival bleeding in smoker and non-smoker patients. Acta Odontologica Scandinavica. 1985 Jan 1;43(5):315-20.
[3] BERGSTRÖM J. Oral hygiene compliance and gingivitis expression in cigarette smokers. European Journal of Oral Sciences. 1990 Dec;98(6):497-503.
[4] Preber H, Bergström J. Effect of cigarette smoking on periodontal healing following surgical therapy. Journal of clinical periodontology. 1990 May;17(5):324-8.
[5] Bergström J, Preber H. Tobacco use as a risk factor. Journal of periodontology. 1994 May 1;65(5):545-50.
[6] Bergstrom, J. (1989) Cigarette smoking as a risk factor in chronic periodontal disease. Community Dentistry and Oral Epidemiology 17, 245–247.
[7] Van Winkelhoff AJ, Bosch-Tijhof CJ, Winkel EG, Van der Reijden WA. Smoking affects the subgingival microflora in periodontitis. Journal of periodontology. 2001 May 1;72(5):666-71.
[8] Haber J. Smoking is a major risk factor for periodontitis. Current opinion in periodontology. 1994:12-8.